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Oxidative stress and cholinesterase inhibition in saliva and plasma of rats following subchronic exposure to malathion

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Abstract

The aim of this study was to examine whether malathion, a commonly used organophosphate (OP), might induce oxidative stress and cholinesterase (ChE) depression in saliva and plasma in rats following subchronic exposure mimicking human exposure. Malathion was administered orally at doses of 100, 500 and 1500 ppm for 4 weeks. Oxidative stress was determined by measuring the malondialdehyde concentration, the end product of lipid peroxidation, and assessing total antioxidant power. Four weeks oral administration of malathion at doses of 100 ppm, 500 ppm and 1500 ppm depressed plasma ChE activity to 45% ($P<0.01$), 48% ($P<0.01$) and 41% ($P<0.01$) of control, respectively. Malathion at doses of 100 ppm, 500 ppm and 1500 ppm depressed saliva ChE activity to 73% ($P<0.01$), 75% ($P<0.01$) and 78% ($P<0.01$) of control, respectively. Malathion at doses of 100 ppm, 500 ppm and 1500 ppm increased plasma antioxidant power by 33% ($P<0.01$), 59% ($P<0.01$) and 118% ($P<0.01$) of control, respectively. Malathion did not change saliva antioxidant power. Malathion at doses of 100 ppm, 500 ppm and 1500 ppm increased plasma thiobarbituric acid reactive substances (TBARS) by 61% ($P<0.01$), 69% ($P<0.01$) and 63% ($P<0.01$) of control, respectively. Malathion at doses of 500 ppm and 1500 ppm increased saliva TBARS by 19% ($P<0.01$) and 22% ($P<0.01$) of control, respectively. Malathion (100 ppm) did not change saliva TBARS level. We concluded that in OP subchronic exposure, depression of ChE is accompanied by induction of oxidative stress that might be beneficial in monitoring OP toxicity.

Keywords: Pesticide; Oxidative stress; Cholinesterase; Saliva; Animal

1. Introduction

Pesticides are used extensively in agriculture to enhance food production by eradicating unwanted insects and controlling disease vectors. The widespread use of pesticides in public health and agricultural programs has caused severe environmental pollution and potential health hazards including severe acute and chronic cases of human poisonings (Abdollahi et al., 1995a,b, 1997; Ellenhorn et al., 1997; Abdollahi et al., 1999; Jalali et al., 2000; Moghadamnia and Abdollahi, 2002; Pajoumand et al., 2002). WHO estimates that the incidence of pesticide poisonings in developing countries has doubled during the past 10 years. It was estimated in 1982 that while developing countries accounted for only 15% of the worldwide use of pesticides, over 50% of pesticide poisoning cases occurred in these countries due to misuse (WHO, 1997). The implications of pesticide resi-

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